Focal Segmental Glomerulosclerosis "FSGS"

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Outlines

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- Aetiological Classification
- Epidemiology
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- Diagnosis and Differential Diagnosis
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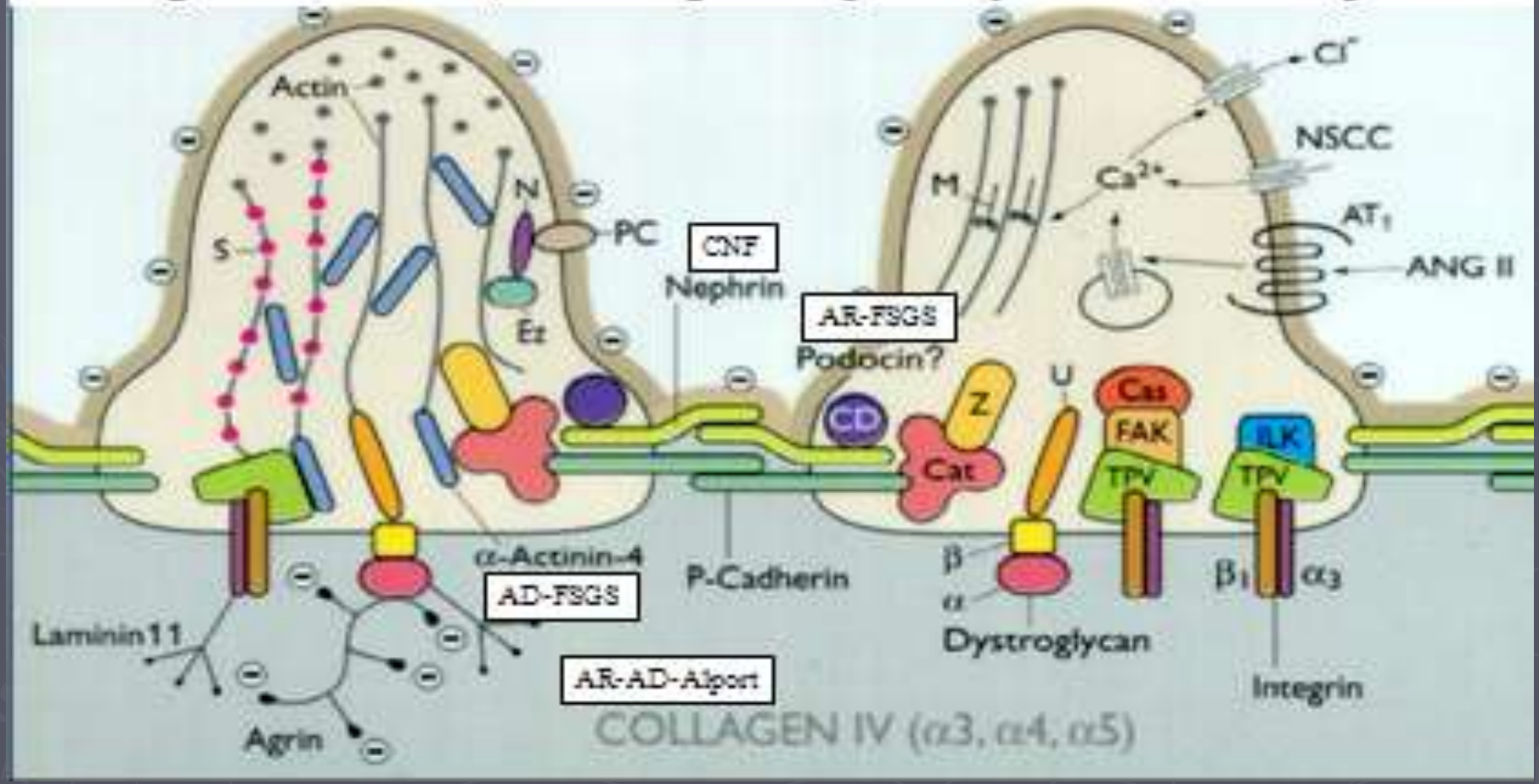
Introduction

- **FSGS** is classified as **idiopathic (primary) FSGS** or **secondary FSGS**.
- **Idiopathic FSGS** is defined by exclusion of any other identifiable cause of secondary FSGS.
- **Secondary FSGS** should be evaluated by:

  Detailed examination of the patient; including **medical history, physical examination, family history, kidney imaging, and kidney pathology; including electron microscopy studies**.
- Although it accounts for only a small percentage of cases of idiopathic nephrotic syndrome in **young children**, **FSGS** represents as many as **35%** of cases in **adults**.
- It is a major cause of progressive renal disease and end-stage renal disease.

Ultrastructure of the slit diaphragm

Cat= Catenin; CD=CD2 associated protein; Ez = ezrin; FAK= focal adhesion kinase; ILK= ntegrin associated kinase; M=myosin; PC= podocalycin; S= synaptopodin; TPV= talin, paxillin, vinculin; U= utrophin; Z=Z-1; FSGS= focal segmental glomerulosclerosis; CNF=congenital nephrotic syndrome of finnish tipe



Etiologic Classification of Focal Segmental Glomerulosclerosis

Primary (Idiopathic) FSGS

Probably mediated by circulating/permeability factor(s)

Secondary FSGS

1. Familial/Genetic*

- Mutations in nephrin *(NPHS1)*
- Mutations in podocin *(NPHS2)*
- Mutations in α-actinin 4 *(ACTN4)*
- Mutations in transient receptor potential cation channel *(TRPC6)*
- Mutations in Wilms tumor suppressor *(WT1)*
- Mutations in inverted formin-2 *(INF2)*
- Mutations in phospholipase C epsilon 1 *(PLCE1)*
- Risk alleles for apolipoprotein L1 *(APOL1)*

2. Virus Associated

- HIV-1 ("HIV-associated nephropathy")
- Parvovirus B19
- Simian virus 40 (SV40)
- Cytomegalovirus (CMV)

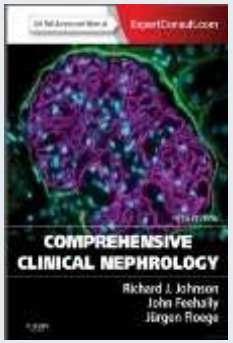
3. Drug Induced

- Heroin ("heroin-nephropathy")
- Interferon
- Lithium
- Pamidronate
- Sirolimus
- Anabolic steroids

4. Mediated by Adaptive Structural-Functional Responses

Reduced renal mass

- Oligomeganephronia
- Very low birth weight
- Unilateral renal agenesis
- Renal dysplasia
- Reflux nephropathy
- Sequela to cortical necrosis
- Surgical renal ablation
- Chronic allograft nephropathy
- Any advanced renal disease with reduction in functioning nephrons



Initially normal renal mass

- Hypertension
- Atheroemboli or other acute vaso-occlusive processes
- Obesity
- Increased lean body mass
- Cyanotic congenital heart disease
- Sickle cell anemia

Epidemiology

- Primary FSGS is slightly more common in **males** than in **females**, and the incidence of ESRD due to FSGS in males of all races is 1.5 to 2 times higher than in females.
- In some countries, such as **Brazil**, FSGS is currently the most common primary renal disease.
- The incidence in both children and adults is higher in **blacks** than in **Caucasians**.
- In the **United States**, FSGS is the most common cause of idiopathic nephrotic syndrome in adult **African Americans**.

  African Americans had a 4 folds greater risk of ESRD from FSGS than **Caucasians** did.

Clinical Manifestations

- Patients with **primary** FSGS present with **asymptomatic proteinuria** or full **nephrotic syndrome**.
- In **children**, 10% to 30% of patients with asymptomatic proteinuria are detected on routine checkups and sports physical examinations.
- In **adults**, asymptomatic detection occurs at military induction examinations, obstetric checkups, and insurance or employment physical examinations.
- The incidence of nephrotic-range proteinuria at onset in **children** is 70% to 90%, whereas 50% to 70% in **adults.**
- **Hypertension** is found in 30% to 50% of children and adults with FSGS at diagnosis.

Clinical Manifestations

- **Microhematuria** is found in 25% to 75% of these patients.
- **A decreased GFR** is noted at presentation in 20% to 30%.
- **Daily urinary protein excretion** ranges from less than **1** to > **30** g/day.

  Proteinuria is typically non-selective.
- **Complement** levels and other **serologic test** results are normal.
- Occasional patients will have **glycosuria**, **aminoaciduria**, **phosphaturia**, or a **concentrating defect** indicating functional tubular damage.

Diagnosis and Differential Diagnosis

- The defining glomerular lesion of FSGS is **focal** and may be confined to **deeper juxtamedullary glomeruli early** in the disease, it may **NOT** be sampled on renal biopsy.
- The **primary** form must be distinguished from **secondary** forms and in general, many forms of adaptive FSGS have lower levels of proteinuria than primary FSGS, a lower incidence of hypoalbuminemia, and on biopsy, lesser degrees of foot process effacement.
- In patients **< 25** years and in those with a family history of FSGS, **genetic screening** for mutations in podocin, nephrin, or other podocyte genes may be useful.

Diagnosis and Differential Diagnosis

- Patients with **FSGS** may be confused with any patient who has **glomerular disease or nephrotic syndrome with negative serologic test results**.
- In **children** with **FSGS**, most of whom present with nephrotic syndrome, the major differential will be between **MCD** and other **variants of steroid-resistant nephrotic syndrome**.
- In **adults** with **subnephrotic proteinuria**, the differential includes almost all **glomerular diseases negative serologic results**.
- In **adults** with **nephrotic syndrome**, **membranous nephropathy and MCD** may present in an identical manner, and only a **renal biopsy** will clarify the diagnosis.

Pathology

- The classical description of FSGS includes **segmental increase of mesangial matrix** with **obliteration** of the **capillaries, sclerosis, hyalinosis, foam cells,** and **segmental scarring,** and **adhesion between the glomerular tuft** and **Bowman's capsule.**
- Early in the disease process, the pattern of glomerulosclerosis is **focal**, involving a minority of glomeruli, and **segmental**, involving a portion of the glomerular tuft.
- Alterations of **podocyte cytoarchitecture** identified on electron microscopy are relatively diffuse, **underscoring** the pathogenetic importance of podocyte injury.
- As the disease progresses, more **diffuse** and **global** glomerulosclerosis evolves.

Pathology

- A classification of FSGS by histologic variants can be applied to both **primary** and **secondary** forms of FSGS.

- Subtypes include **classic**, **or Not otherwise specified "NOS"**; **perihilar variant**, in which more than 50% of glomeruli with segmental lesions involving the vascular pole region; **cellular variant**, manifesting endocapillary hypercellularity; **collapsing variant**, in which at least one glomerulus has global collapse and overlying visceral cell hypertrophy and hyperplasia; and **tip variant**, with segmental lesions involving the tubular pole.

I- Classic Focal Segmental Glomerulosclerosis (FSGS Not Otherwise Specified "NOS")

- This is the **most frequent** variant and may occur in primary or secondary forms of FSGS.
- It is defined by **accumulations of extracellular matrix that occlude glomerular capillaries, forming discrete segmental solidifications.**
- There may be **hyalinosis** (plasmatic insudation of amorphous glassy material beneath the GBM), **endocapillary foam cells.**
- **Adhesions to Bowman capsule** are common, and overlying visceral epithelial cells often appear swollen and form a cellular "**cap**" over the sclerosing segment.
- **Tubular atrophy and interstitial fibrosis** are commensurate with the degree of glomerulosclerosis.

Classic Focal Segmental Glomerulosclerosis (FSGS Not Otherwise Specified)

- **Immunofluorescence** typically reveals focal and segmental granular deposition of **IgM, C3**, and more variably **C1** in the distribution of the segmental glomerular sclerosis.
- **Electron Microscopy**; segmental sclerotic lesions exhibit **increased ECM**, **wrinkling** and **retraction** of the GBM, **accumulation of inframembranous hyaline**, and resulting **narrowing or occlusion of the glomerular capillary lumina.**

  The adjacent nonsclerotic glomerular capillaries show **foot process effacement** with variable microvillous transformation

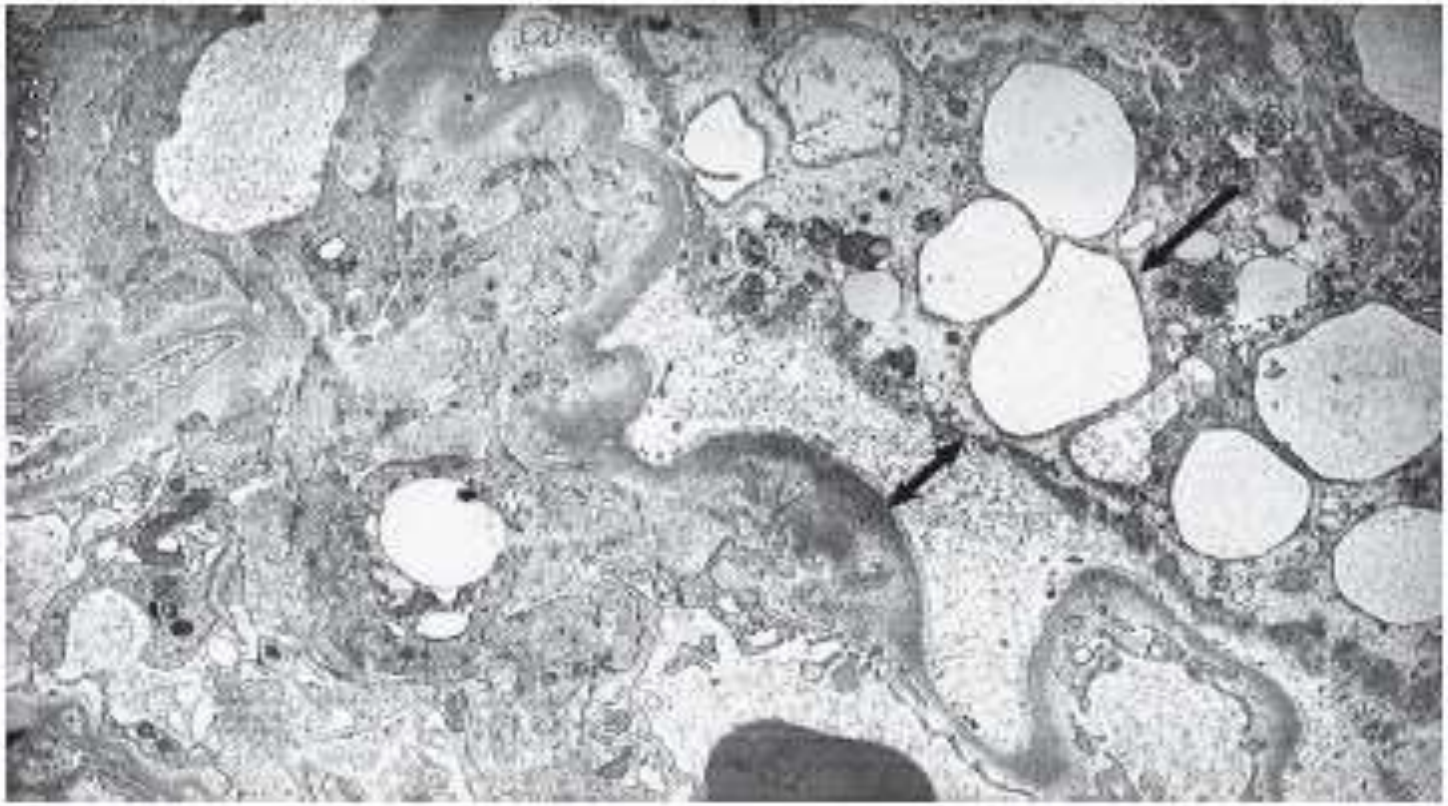


Figure 18-4 Focal segmental glomerulosclerosis, not otherwise specified. Electron micrograph illustrates the lesion of segmental sclerosis with obliteration of the glomerular capillaries by increased extracellular matrix with wrinkled and retracted glomerular basement membranes. The overlying podocytes are detached, with complete effacement of foot processes *(double-headed arrow)* and numerous electron-lucent intracellular transport vesicles *(arrow)*. (×2500.)

II- Perihilar Variant of Focal Segmental Glomerulosclerosis

- The **perihilar** variant is defined as perihilar hyalinosis and sclerosis involving more than 50% of glomeruli with segmental lesions.
- Although the **perihilar** variant may occur in **primary** FSGS, it is particularly frequent in **secondary** forms of FSGS mediated by **adaptive structural-functional responses**, where it is typically accompanied by **glomerular hypertrophy** (**glomerulomegaly**) and has relatively **mild foot process effacement.**
- In this setting, reflex dilation of the **afferent arteriole** and the greater filtration pressures at the proximal end of the glomerular capillary bed may favor the development of lesions at the **vascular pole**.

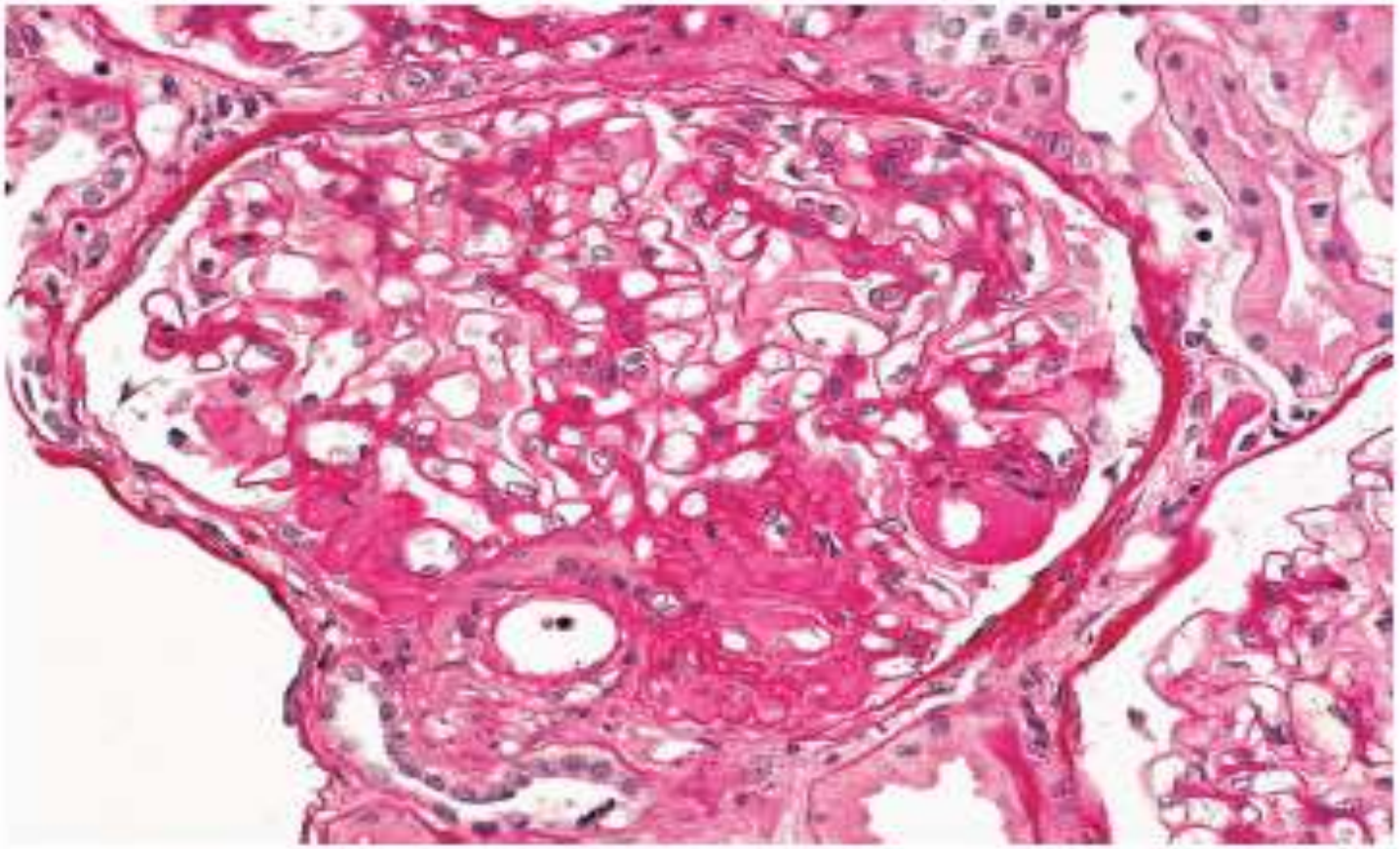


Figure 18-5 Focal segmental glomerulosderosis (FSGS), perihilar variant. A discrete lesion of segmental sclerosis and hyalinosis is located at the glomerular vascular pole (i.e., perihilar). The glomerulus is hypertrophied. The patient had secondary FSGS in the setting of solitary kidney as a result of contralateral renal agenesis. (PAS; ×250.)

III- Cellular Variant of Focal Segmental Glomerulosclerosis

- The **cellular** variant is characterized by focal and segmental **endocapillary hypercellularity** that may mimic a form of focal proliferative glomerulonephritis.
- Glomerular capillaries are segmentally occluded by endocapillary hypercellularity, including **foam cells**, **infiltrating leukocytes**, **karyorrhectic debris**, and **hyaline**.
- There is often **hyperplasia** of the **visceral epithelial cells**, which may appear swollen and crowded, sometimes forming **pseudocrescents**.
- **Foot process effacement** is typically severe.
- Cellular FSGS is thought to represent an early stage in the development of segmental lesions and is usually **primary**.

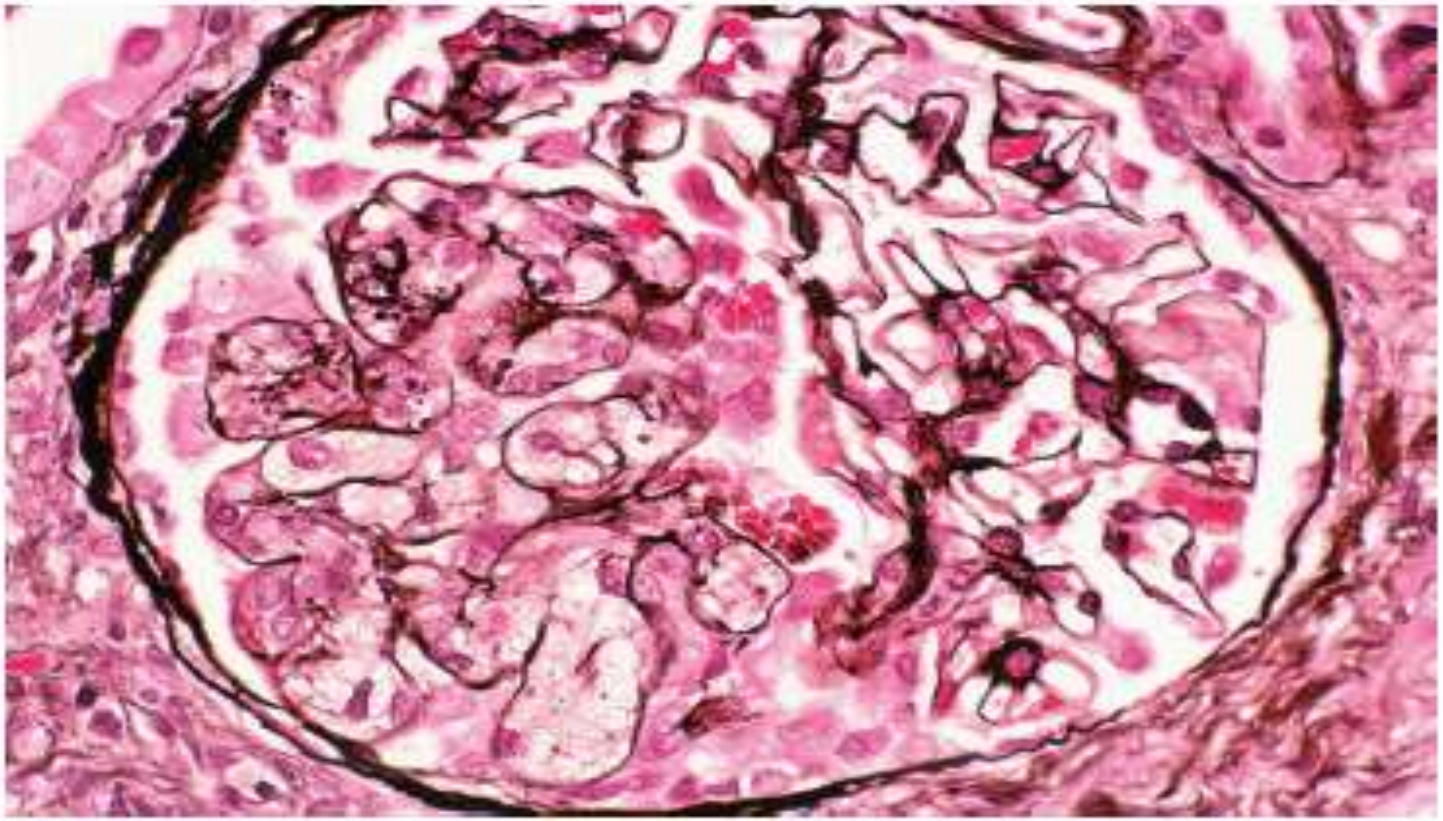


Figure 18-6 Focal segmental glomerulosclerosis, cellular variant. The glomerular capillary lumina are segmentally occluded by endocapillary cells, including foam cells, infiltrating mononuclear leukocytes, and pyknotic debris. The findings mimic a proliferative glomerulonephritis because of the hypercellularity and absence of extracellular matrix material. There are hypertrophy and hyperplasia of the overlying visceral epithelial cells, some of which contain protein resorption droplets. (Jones methenamine silver; ×400.)

IV- Collapsing Variant of Focal Segmental Glomerulosclerosis

- The collapsing variant is defined by at least one glomerulus with **segmental or global collapse** and overlying hypertrophy and hyperplasia of visceral epithelial cells.
- In these areas, there is occlusion of glomerular capillary lumina by implosive wrinkling and GBM collapse**. The collapsing lesion is more often global than segmental.**
- Glomerular epithelial cells often contain prominent intracytoplasmic protein resorption droplets and may fill Bowman space, forming **pseudocrescents**.
- In collapsing FSGS, there is prominent **tubulointerstitial disease**, including **tubular atrophy**, **interstitial fibrosis**, **interstitial edema**, and **inflammation**.
- On **EM**, there is typically **severe foot process effacement** affecting both collapsed and noncollapsed glomeruli.
- Collapsing glomerulopathy may occur as **1ry** or **2ry** form of FSGS.

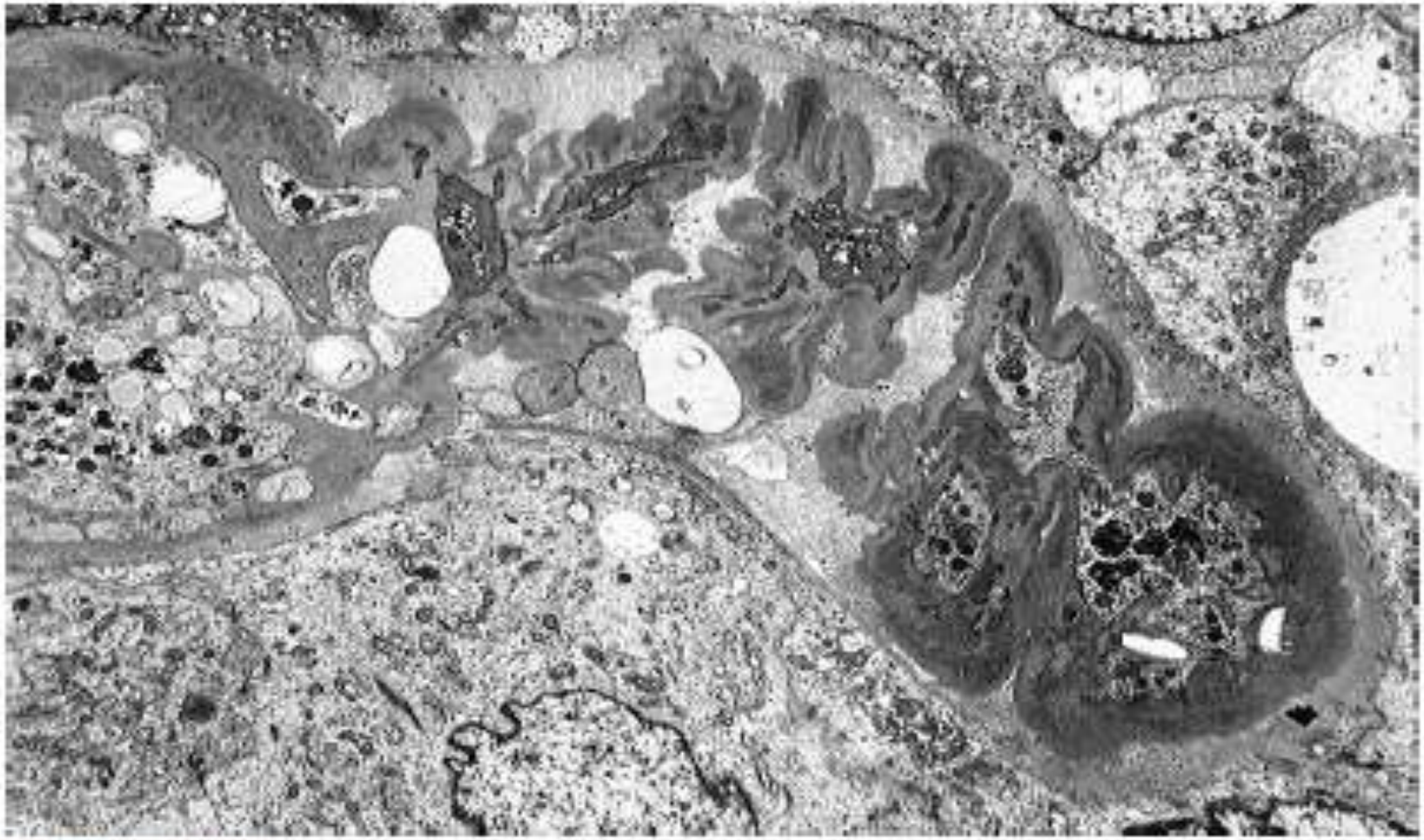


Figure 18-10 Focal segmental glomerulosclerosis, collapsing variant. On electron microscopy, there is tight collapse of the glomerular capillaries with corrugated glomerular basement membrane. The overlying podocytes appear detached and hypertrophied, with complete loss of foot processes. (×2500.)

V- Tip Variant of Focal Segmental Glomerulosclerosis

- The **tip variant** is defined by the presence of at least one segmental lesion involving the **tip** domain (the outer 25% of the tuft next to the origin of **the proximal tubule**).
- There is either **adhesion** between the tuft and Bowman capsule or confluence of swollen podocytes with parietal or tubular epithelial cells at **the tubular lumen or neck**.
- The segmental lesions may be **cellular** or **sclerosing.**
- The **foot process effacement is usually severe**.
- Most cases are **primary** and resemble **MCD** in the abrupt onset of nephrotic syndrome, suggesting they may share a similar permeability factor.
- Higher shear stress and tuft prolapse at the tubular pole are likely to play a role in the morphogenesis of this lesion.

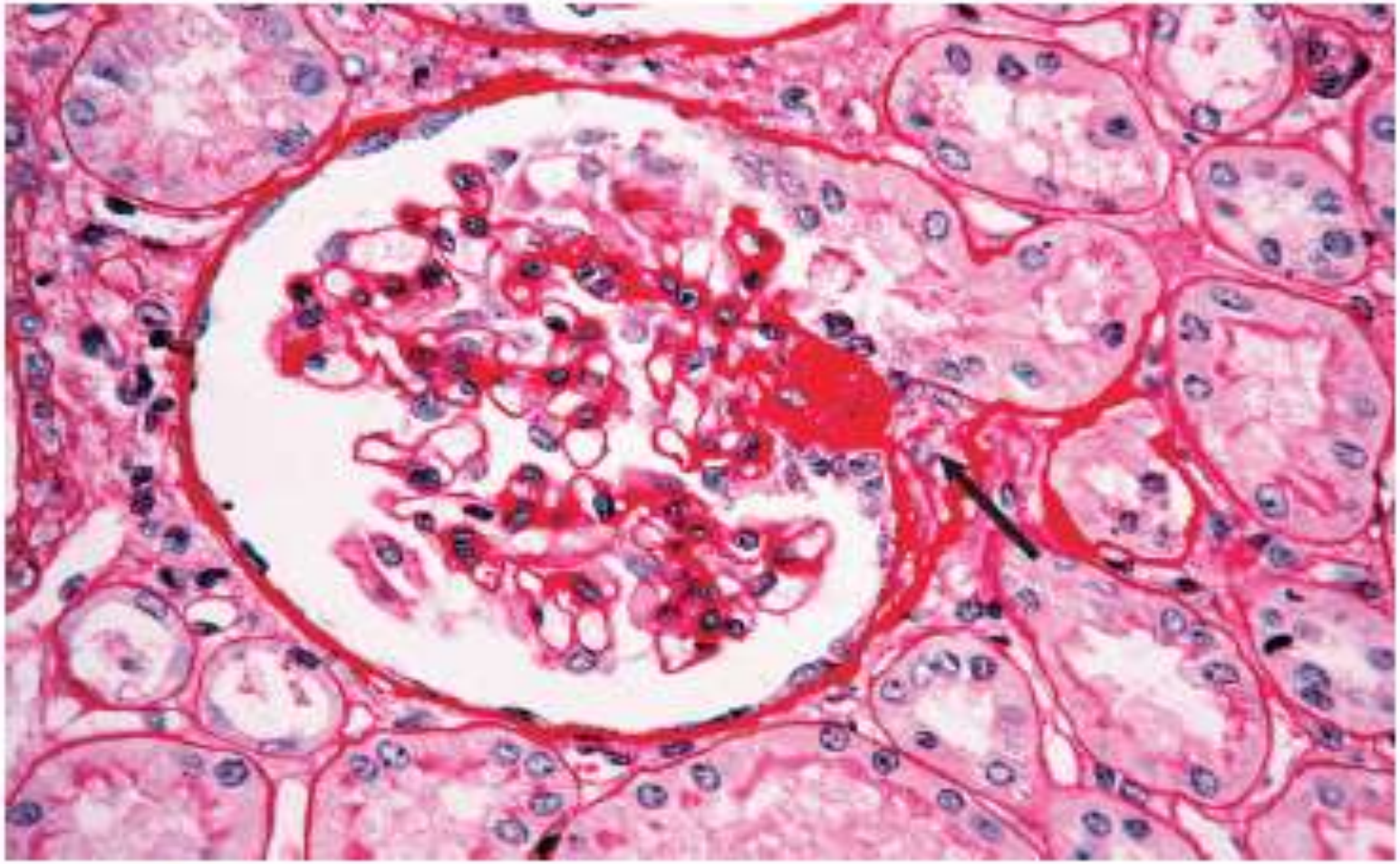


Figure 18-13 **Focal segmental glomerulosclerosis, tip lesion variant.** A sclerosing tip lesion forms an adhesion to the tubular pole *(arrow)*. (PAS; ×250.)

Risk Factors for Progressive Renal Disease in Focal Segmental Glomerulosclerosis

Clinical Features at Biopsy

Severity of nephrotic-range proteinuria

Elevated serum creatinine

Black race

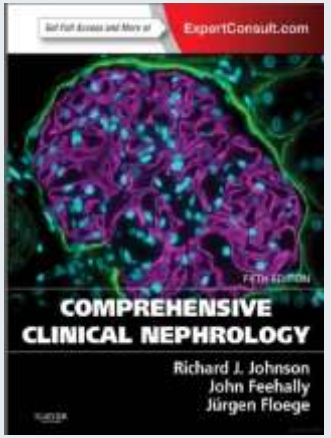
Histopathologic Features at Biopsy

Collapsing variant

Tubulointerstitial fibrosis

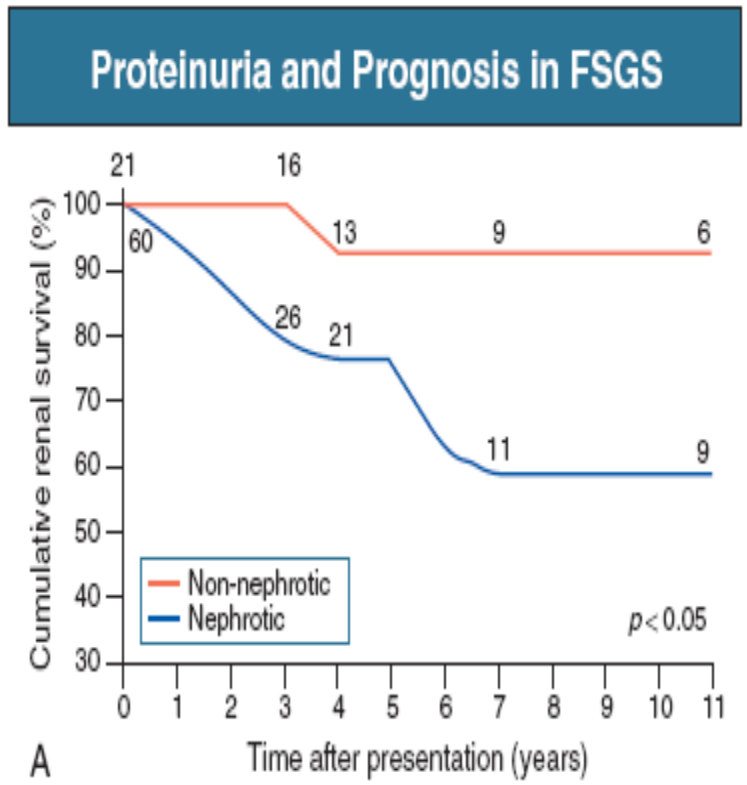
Clinical Features During Disease Course

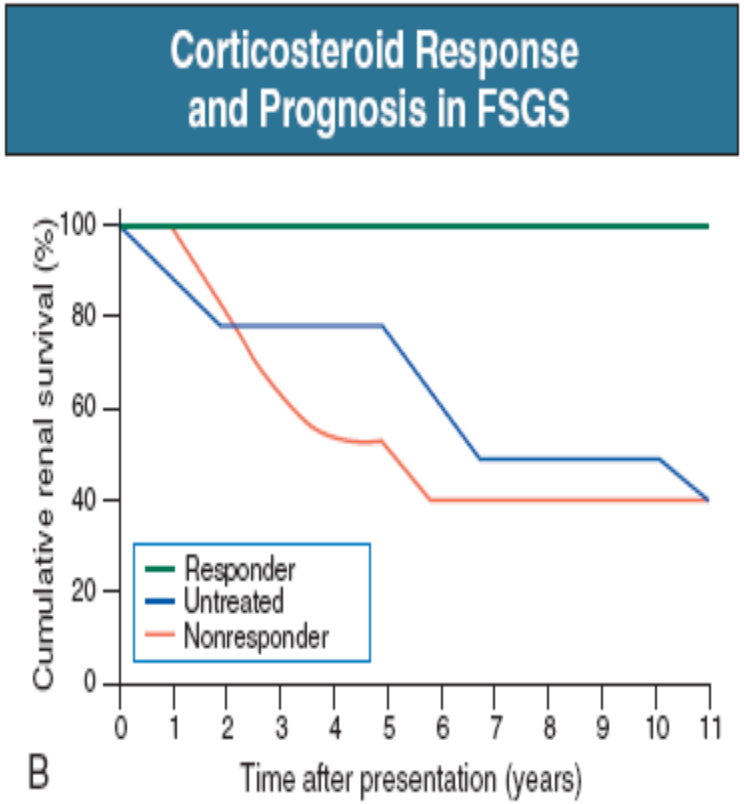
Failure to achieve partial or complete remission



Box 18-3 Risk factors for progressive renal disease in FSGS.

NATURAL HISTORY AND PROGNOSIS





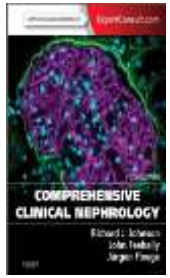


Figure 18-18 **Prognosis in primary focal segmental glomerulosclerosis (FSGS). A,** The risk for development of renal failure is related to the extent of proteinuria. Those with nephrotic-range proteinuria are much more likely to develop renal failure than those with low-grade proteinuria. The figures indicate the number of at-risk patients at different time points. **B,** Corticosteroid-responsive patients are significantly less likely to develop renal failure than nonresponders and untreated patients. *(Modified from reference 87.)*

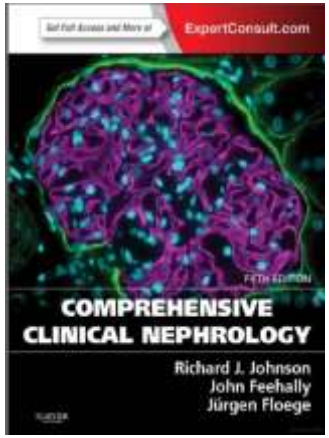
Treatment

- The potential benefit of therapy includes disease ***cure, control, and/or slowing the progression to ESRD***.
- In FSGS, outcome parameters can be divided into **kidney** and **proteinuric** events. Disease cure and control are defined primarily by changes in proteinuria.
- In most cases of idiopathic FSGS, the natural history of the disease is prolonged, with even **complete remitters** having a **relapse rate** of up to **40%**.
- There is also a significant minority with **NO** response to therapy; hence, the potential benefits of treatment must be constantly **weighed** against the risks of the chosen immunosuppressive therapy.

Definitions of nephrotic syndrome in adults with FSGS

Classification	Definition
Complete remission	Reduction of proteinuria to <0.3 g/d or <300 mg/g (<30 mg/mmol), urine creatinine and normal serum creatinine and serum albumin >3.5 g/dl (35 g/l)
Partial remission[a]	Reduction of proteinuria to 0.3–3.5 g/d (300–3500 mg/g [30–350 mg/mmol]), urine creatinine and stable serum creatinine (change in creatinine <25%) or Reduction of proteinuria to 0.3–3.5 g/d (300–3500 mg/g [30–350 mg/mmol]), urine creatinine and a decrease >50% from baseline, and stable serum creatinine (change in creatinine <25%)
Relapse	Proteinuria >3.5 g/d or >3500 mg/g (>350 mg/mmol) urine creatinine after complete remission has been obtained
Frequent relapse	Not defined in adults
Steroid-dependent	Two relapses during or within 2 weeks of completing steroid therapy
Steroid-resistant	Persistence of proteinuria despite prednisone 1 mg/kg/d or 2 mg/kg every other day for >4 months

FSGS, focal segmental glomerulosclerosis; GFR, glomerular filtration rate.
[a]Both definitions of partial remission have been used in the literature.



Therapeutic Options in FSGS

- Subnephrotic proteinuria without symptoms →
- Nephrotic syndrome, symptomatic, high risk of complications of nephrotic syndrome →
- Alternative therapy for steroid-resistant patients or for patients with contraindications to steroid therapy →

Figure 18-19 Therapeutic options in focal segmental glomerulosclerosis. Treatment of secondary FSGS should be directed at the underlying cause whenever possible. For HIV-associated nephropathy, treatment with highly active antiretroviral therapy (HAART); for pamidronate nephrotoxicity, discontinue the medication; and for obesity-related glomerulopathy, weight loss. *ACE*, Angiotensin-converting enzyme; *ARB*, angiotensin receptor blocker; *BP*, blood pressure; *MMF*, mycophenolate mofetil.

Treatment

6.2: Initial treatment of FSGS

6.2.1: We recommend that corticosteroid and immunosuppressive therapy be considered only in idiopathic FSGS associated with clinical features of the nephrotic syndrome. **(1C)**

6.2.2: We suggest prednisone be given at a daily single dose of 1 mg/kg (maximum 80 mg) or alternate-day dose of 2 mg/kg (maximum 120 mg). **(2C)**

6.2.3: We suggest the initial high dose of corticosteroids be given for a minimum of 4 weeks; continue high-dose corticosteroids up to a maximum of 16 weeks, as tolerated, or until complete remission has been achieved, whichever is earlier. **(2D)**

6.2.4: We suggest corticosteroids be tapered slowly over a period of 6 months after achieving complete remission. **(2D)**

6.2.5: We suggest CNIs be considered as first-line therapy for patients with relative contraindications or intolerance to high-dose corticosteroids (e.g., uncontrolled diabetes, psychiatric conditions, severe osteoporosis). **(2D)**

Treatment

RAAS Blockade and Blood Pressure Control

- Optimal conservative management of patients with FSGS should follow guidelines for patients with persistent proteinuria .
- RAAS blockade should be routine; however, it may be delayed in nephrotic syndrome to see if there is a response to initial corticosteroid therapy.
- This is particularly relevant if the nephrotic syndrome is severe, since the risk of developing AKI due to hypoperfusion and acute tubular necrosis is increased in this setting.

Treatment

Corticosteroids

- **Corticosteroid** therapy should only be considered for patients with idiopathic FSGS associated with nephrotic syndrome.
- There are **no** data to support treatment with corticosteroids in patients **without nephrotic-range proteinuria.**
- Treatment routines have varied with durations from **4 to 24 months**, and prednisone dosing **from 0.3 to 1.5 mg/kg/d**, reported complete remission rates range from 28% to 74%, and partial remission rates from 0% to 50%.
- The average time to complete remission is 3 – 4 months, with a range up to 8 months.

Treatment

Corticosteroids

- The timing of prednisone therapy initiation has been debated. Spontaneous remissions do occur, with reported rates varying from 5% to 23%.
- Spontaneous remissions are more likely to occur in patients with **tip lesions, with preserved kidney function, and lower grades of proteinuria**. In such patients, prednisone treatment could be delayed to see if spontaneous remission occurs with RAAS blockade and other conservative approaches.
- There is **NO** evidence to support the use of corticosteroids in **secondary FSGS** and, in current practice, such patients are **not** treated with **immunosuppressive therapy.**

Treatment

Other Immunosuppressive Agents

- A retrospective observational study compared **high-dose oral prednisone** (1 mg/kg/d) for at least 4 months and tapering thereafter, with **low-dose prednisone** (0.5 mg/kg/d) in combination with **cyclosporine** (3 mg/kg/d initial dose, tapering to 50 mg/d) or **azathioprine** (2 mg/kg/d initial dose, tapering to 0.5 mg/kg/d).
  - Average duration of treatment was 20 months.
  - Remission rates were comparable; 63% for prednisone (n=9), 80% for prednisone plus azathioprine (n=6), and 86% for prednisone plus cyclosporine (n=10).
- Another study used **tacrolimus** as initial therapy (n=6) and noted a remission in 100 %.

Treatment

Other Immunosuppressive Agents

- A randomized study in adult patients with FSGS and persistent nephrotic syndrome after 6 months of **RAAS** blockade compared **MMF** (2 g/d for 6 months) plus low dose prednisone (0.5 mg/kg/d for 8–12 weeks) to high dose prednisone (1 mg/kg/d for 12–24 weeks, followed by tapering over 8 weeks).

Similar remission rates were observed in the two regimens, 71% (12/17 patients) vs. 69% (11/16 patients).

- A **CNI** is favored in view of the evidence derived from studies in patients with steroid-resistant FSGS.

Treatment

6.3: Treatment for **relapse**

- We suggest that a relapse of nephrotic syndrome is treated as per the recommendations for relapsing MCD in adults:

5.2.1: We suggest **oral cyclophosphamide** 2–2.5mg/kg/d for 8 weeks. **(2C)**

5.2.2: We suggest **CNI** (**cyclosporine** 3–5mg/kg/d or **tacrolimus** 0.05-0.1mg/kg/d in divided doses) for 1–2 years for FR/SD MCD patients who have relapsed despite **cyclophosphamide**, or for people who wish to preserve their fertility. **(2C)**

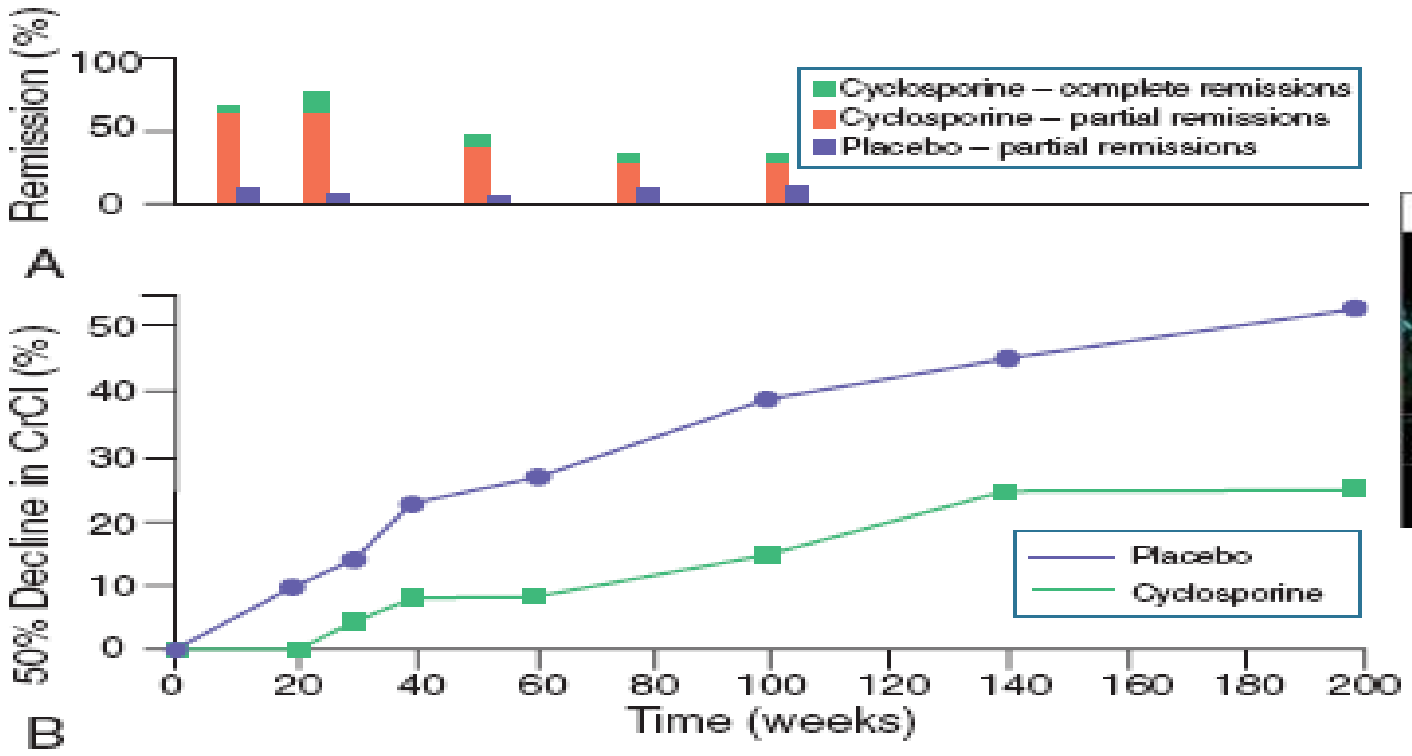
5.2.3: We suggest **MMF** 500 –1000 mg twice daily for 1–2 years for patients who are intolerant of **corticosteroids**, **cyclophosphamide**, and **CNIs**. **(2D)**

Treatment

6.4: Treatment for **steroid-resistant** FSGS

- **6.4.1:** For steroid-resistant FSGS, we suggest that **cyclosporine** at 3–5 mg/kg/d in divided doses be given for at least 4–6 months. **(2B)**
- **6.4.2:** If there is a partial or complete remission, we suggest continuing **cyclosporine** treatment for at least **12 months**, followed by a slow taper. **(2D)**
- **6.4.3:** We suggest that patients with steroid-resistant FSGS, who do not tolerate **cyclosporine**, be treated with a combination of **mycophenolate mofetil** and high-dose **dexamethasone**. **(2C)**

Cyclosporine in Corticosteroid-Resistant FSGS



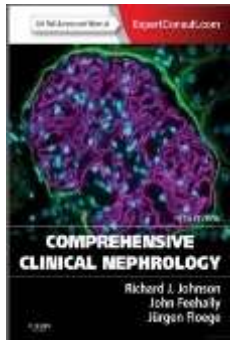


Figure 18-20 Cyclosporine in corticosteroid-resistant focal segmental glomerulosclerosis (FSGS). Randomized controlled trial of 6 months of treatment with prednisolone and either cyclosporine or placebo. **A,** Cyclosporine induces a partial or complete remission significantly more often than placebo. **B,** Cyclosporine treatment results in a lower rate of decline in renal function than with placebo, even after 4 years; *CrCl,* creatinine clearance. *(Modified from reference 70.)*

Treatment

- There is no agreement in the literature regarding the duration of **prednisone** therapy that defines steroid-resistance.
- Some authors advise the use of alternative immunosuppressive therapy after only 4–8 weeks of prednisone, whereas others define resistance as persistent nephrotic syndrome after 4 months prednisone in a dose of 1 mg/kg/d.
- We suggest that **prednisone** be given for 4 months before defining resistance to therapy.

Treatment

RATIONALE

- **Cyclosporine** is effective in inducing remission of proteinuria in patients with steroid-resistant FSGS. Remissions can develop slowly, and may take 3–6 months after start of therapy.
- A partial remission provides a substantial outcome benefit.
- Relapses are very frequent after withdrawal of **cyclosporine**. More prolonged treatment may lead to more persistent remissions. Relapses occur frequently when using **cyclosporine** for a 6-month period.
- A longer duration of therapy and slow tapering strategy in **cyclosporine - responsive** patients can be used in FSGS similar to that advised in adults with MCD.
- There is limited evidence to support the efficacy of other regimens in patients with steroid-resistant proteinuria.

Treatment

Other Immunosuppressive Agents

- A recent RCT compared **cyclosporine** to the combination of **MMF and high-dose dexamethasone** in children and young adults with steroid-resistant FSGS.

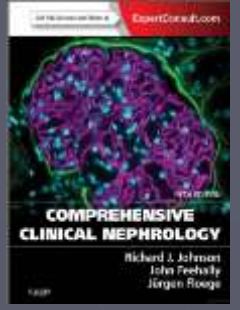
  There was **no** statistically significant difference in remission rates.

  The study was largely underpowered, and inferiority of the **MMF** regimen could not be excluded.
- Case reports and small observational studies have reported response to **alkylating agents, sirolimus, and rituximab**, but there is insufficient evidence to support the use of any of these agents in patients with steroid-resistant FSGS.

Treatment

- **Plasma exchange**, which is successful in treating some patients with recurrent FSGS in the renal allograft, has **NOT** proved useful in patients with disease in their native kidneys.
- A study of **low-density lipoprotein apheresis** and prednisone in corticosteroid and cyclosporine resistant children with primary FSGS achieved a remission in 7 of 11 patients.
- In patients with primary idiopathic or a secondary form of FSGS who remain nephrotic, control of fluid retention and edema can be managed with **salt restriction and diuretics**.

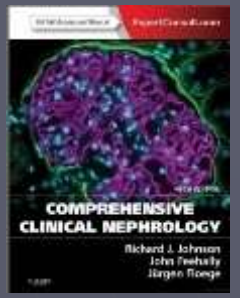
Transplantation



- Approximately **40%** of patients with primary FSGS who develop ESRD and undergo renal transplantation develop recurrent FSGS in the allograft.
- Children with FSGS and patients who manifest with **more severe proteinuria** and a **more rapid course to renal failure** in **their native kidneys** are **at greater risk** of **recurrence** in the allograft.

  Those who have **lost a prior allograft** because of **recurrent FSGS** are at highest risk of recurrence.
- Recurrence in the allograft may be seen **immediately** after transplantation (supporting the existence of a circulating factor) or **years later**.

Transplantation



- Interestingly, the histologic variant of recurrent FSGS was the same as that documented in the native kidney in **81%** of cases, validating the fidelity of the histologic subclassification.
- **Plasma exchange** has been used successfully to induce remissions of the proteinuria associated with recurrence, but the results are more favorable in children than in adults.
- **Rituximab** has been used in some patients for recurrent FSGS, with varied outcomes.

THANK YOU

Dakahlia Nephrology Group
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